aggressive stenting strategy on initial and one-year follow-up costs in patients undergoing coronary angioplasty. Aust N Z J Med 1999;29: 243–8.
31. Hillegas WB, Newman AR, Raco DL. Economic issues in glycoprotein IIb/IIIa receptor therapy. Am Heart J 1999;138:S24–32.
32. Goklaney AK, Murphy JD, Hillegass WB Jr. Abciximab therapy in percutaneous intervention: economic issues in the United States. Am Heart J 1998;135:S90–7.
33. Aristides M, Gliksman M, Rajan N, Davey P. Effectiveness and cost-effectiveness of single bolus treatment with abciximab (Reo Pro) in preventing restenosis following percutaneous transluminal coronary angioplasty in high risk patients. Heart 1998;79:12–7.
34. The TIMI Study Group. Effects of tissue plasminogen activator and a comparison of early invasive and conservative strategies in unstable angina and non-Q-wave myocardial infarction: results of the TIMI-IIIB Trial. Thrombolysis in Myocardial Ischemia. Circulation 1994; 89:1545–56.
35. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Lancet 1994;343:311–22.
36. Rogers WJ, Bowlby LJ, Chandra NC, et al. Treatment of myocardial infarction in the United States (1990 to 1993). Observations from the National Registry of Myocardial Infarction. Circulation 1994;90: 2103–4.
37. Barron HV, Bowlby LJ, Breen T, et al. Use of reperfusion therapy for acute myocardial infarction in the United States: data from the National Registry of Myocardial Infarction 2. Circulation 1998;97: 1150–6.
38. Ohman EM, Lincoff AM, Bode C, et al. Enhanced early reperfusion at 60 minutes with low-dose reteplase combined with full-dose abciximab in acute myocardial infarction: preliminary results from the GUSTO IV (SPEED) dose ranging trial. Circulation 1998;98:I-504.
39. Antman EM, Guigliano RP, Gibson M, et al. Abciximab facilitates the rate and extent of thrombolysis: results of the Thrombolysis in Myocardial Infarction (TIMI) 14 trial. Circulation 1999;99:2720–32.
40. Weaver WD, Simes J, Betriu A, et al. Comparison of primary coronary angioplasty and intravenous thrombolytic therapy for acute myocardial infarction. JAMA 1997;278:2093–8.
41. Brodie BR, Stuckey TD, Wall TC, et al. Importance of time to reperfusion for 30-day and late survival and recovery of left ventricular function after primary angioplasty for acute myocardial infarction. J Am Coll Cardiol 1998;32:1312–9.
42. Kennedy JW, Stadius ML. Combined thrombolytic and platelet glycoprotein IIb/IIIa inhibitor therapy for acute myocardial infarction: will pharmacological therapy ever equal primary angioplasty? Circulation 1999;99:2714–6.
43. The GUSTO Angiographic Investigators. The effects of tissue plasminogen activator, streptokinase, or both on coronary-artery patency, ventricular function, and survival after acute myocardial infarction. N Engl J Med 1993;329:1615–22.
44. O'Neill WW, Brodie BR, Ivanhoe R, et al. Primary coronary angioplasty for acute myocardial infarction (The Primary Angioplasty Registry). Am J Cardiol 1994;73:627–34.
45. Zijlstra F, Beukema WP, van't Hof AW, et al. Randomized comparison of primary coronary angioplasty with thrombolytic therapy in low risk patients with acute myocardial infarction. J Am Coll Cardiol 1997;29:908–12.
46. Grines CL, Browne KF, Marco J, et al. A comparison of immediate angioplasty with thrombolytic therapy for acute myocardial infarction: the Primary Angioplasty in Myocardial Infarction Study Group. N Engl J Med 1993;328:673–9.
47. Zijlstra F, Jan de Beer M, Hoorntje JCA, et al. A comparison of immediate coronary angioplasty with intravenous streptokinase in acute myocardial infarction. N Engl J Med 1993;328:680–4.
48. Gibbons RJ, Holmes DR Jr, Reeder GS, et al. Immediate angioplasty compared with the administration of a thrombolytic agent followed by conservative treatment for myocardial infarction. N Engl J Med 1993;328:685–91.
49. Grines CL, Marsalese DL, Brodie B, et al. Safety and cost-effectiveness of early discharge after primary angioplasty in low risk patients with acute myocardial infarction. PAMI-II Investigators. Primary Angioplasty in Myocardial Infarction. J Am Coll Cardiol 1998;31:967–72.
50. Garcia E, Elizaga J, Perez-Castellano N, et al. Primary angioplasty versus systemic thrombolysis in anterior myocardial infarction. J Am Coll Cardiol 1999;33:605–11.
51. Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. N Engl J Med 1999;341:625–34.

Nonocclusive Coronary Dissections: To Stent or Not to Stent?

Cappelletti et al. (1) reviewed the outcome of 129 consecutive patients treated with conventional angioplasty (PTCA) at a time when coronary stents were not available. Patients (45; 35%) presenting nonocclusive dissections post-PTCA had a significantly lower restenosis rate than patients without dissections (12% vs. 44%; $p < 0.001$). The restenosis rate in another group of patients undergoing stenting for nonocclusive dissection (clinically and angiographically matched), later on in their experience, was 25% (1). At a time when coronary stenting is experiencing an exponential increase, these results would appear rather provocative. Some classical studies, however, also suggested that most dissections are not only benign but also predict a lower restenosis rate (2,3). Nevertheless, no previous study was able to demonstrate such a low restenosis rate in this cohort of patients.

Furthermore, to demonstrate convincingly that a conservative approach—namely a "watchful waiting" strategy—may even be superior in the long run to coronary stenting is much more challenging. Given the potential clinical implications of this study, some methodological clarifications appear warranted.

First, it is not clear why two patients with vessel closure were excluded. Keeping in mind that this is a retrospective study, it will be important to know whether these dissections were flow-limiting immediately after PTCA or flow deterioration occurred later on. Second, 67% (33/49) of the nontreated dissections were type A versus none (0/60) of the stented dissections (chi-square $p < 0.0001$). Therefore, it is difficult to assume that these two populations were similar, and thus direct comparison of results may not be appropriate. Further details on whether the restenosis rate tended to cluster around patients with type C-D dissections (untreated/stented groups) will be helpful.

Finally, the methodology of quantitative coronary analysis was not specified. This is relevant because the analysis of dissected coronary segments is technically demanding. In fact, at first glance it appears difficult to explain a mean lumen diameter post-PTCA of 3.23 ± 0.65 mm (reference 3.20 ± 0.54 mm) yielding a $20 \pm 7\%$ diameter stenosis. The large lumen diameter of the dissected segments indicates that the dissection image was fully included into the lumen measurements. This is in contradistinction with some prior studies using careful edge-detection quantitative angiography (4,5). We previously demonstrated (5) that residual coronary dissections after stenting had a benign outcome when they were stable, were not associated with significant lumen narrowing, and did not compromise coronary flow.

Our data (5) also concur with the current study, suggesting that most residual dissections disappear at follow-up. Moreover, these dissected coronary segments may promote a unique pattern of vessel remodeling that could explain a lower restenosis rate (1) or even a significant lumen improvement on late angiography (5). We fully agree with the idea that conservative management of coronary

dissections is attractive in selected cases (adverse anatomy, small vessels, type A-B dissections). However, we believe that only properly designed studies will be able to determine whether this strategy is superior to stenting in most patients experiencing nonocclusive dissections. In the interim, accepting the potential risk of vessel closure and the logistic implications (prolonged observation or even repeat angiography) inherently associated with the conservative strategy should be weighted against the results of coronary stenting using currently available stent designs. Although we sympathize with the words of caution against the indiscriminate use of stents, it would appear more reasonable to challenge first the systematic use of "elective" stenting in clinical/angiographic settings where its efficacy—as compared with PTCA—remains largely unsettled.

Fernando Alfonso, MD, PhD, FESC
Interventional Cardiology Department
San Carlos University Hospital
Madrid, Spain

PII S0735-1097(00)00670-7

REFERENCES

1. Cappelletti A, Margonato A, Rosano G, et al. Short- and long-term evolution of unstented nonocclusive coronary dissection after coronary angioplasty. J Am Coll Cardiol 1999;34:1484–8.
2. Leingruber PP, Roubin GS, Anderson HV, et al. Influence of intimal dissections on restenosis after successful coronary angioplasty. Circulation 1985;72:530–50.
3. Ellis SG, Roubin GS, King SB, Douglas JS, Cox WR. Importance of stenosis morphology in the estimation of restenosis risk after elective percutaneous transluminal coronary angioplasty. Am J Cardiol 1989;63:30–4.
4. Ozaki Y, Violaris AG, Kobayashi T, et al. Comparison of coronary luminal quantification obtained from intracoronary ultrasound and both geometric and videodensitometric quantitative angiography before and after balloon angioplasty and atherectomy. Circulation 1997;96:491–9.
5. Alfonso F, Hernandez R, Goicolea J, et al. Coronary stenting for acute coronary dissection after coronary angioplasty: implications of residual dissection. J Am Coll Cardiol 1994;24:989–95.

REPLY

Dr. Alfonso asks why two patients in our study (1) with occlusive dissection after percutaneous transluminal coronary angioplasty (PTCA) were excluded and when these dissections occurred. As it is clearly stated in the article these two type E dissections evolved toward complete artery occlusion during the procedure and how they caused an acute myocardial infarction immediately after the procedure. Because the study reported the results of nonocclusive unstented dissections, they were excluded from the analysis at the beginning.

As far as the second point is concerned, we have acknowledged the higher prevalence of lesions A and B in the unstented group, but this limitation derives from the later stage in which the stented patients were assessed, when the easy availability of stenting allowed higher inflation pressures. However, although unstented patients had a higher prevalence of dissections grades A and B (namely 85% vs. 56% at 24 h), the restenosis rate for stented and unstented patients was similar for each dissection grade (p = NS).

What we would like to stress in our study is that in this stenting era, where there is a growing and widespread use of these devices (2), the "minor" dissections (type A and B), most frequently occurring during PTCA, are associated with a very low risk of complications and restenosis, suggesting a more conservative approach.

Finally, Dr. Alfonso states that "the large lumen diameter of the dissected segments indicates that the dissection image was fully included into the lumen measurements." However, as clearly shown in Table 1 of our article, the mean lumen diameter post-PTCA in dissected vessels was not 3.23 ± 0.65 mm but 3.11 ± 0.89 mm, a lower value than that of the mean reference artery diameter pre-PTCA (3.18 ± 0.7 mm) in the same vessels. We do agree that the methodology of quantitative coronary angiography is technically demanding, especially for the analysis of dissected segments. Therefore, we are promoting in our Institute new and different tools for quantitative analysis, such as intracoronary ultrasound (IVUS), coronary Doppler evaluation, and myocardial fractional flow-reserve measurement.

Alberto Cappelletti, MD
Alberto Margonato, MD
Division of Cardiology
Istituto Scientifico H.S. Raffaele
Milan, Italy

PII S0735-1097(00)00669-0

REFERENCES

1. Cappelletti A, Margonato A, Rosano G, et al. Short- and long-term evaluation of unstended nonocclusive coronary dissection after coronary angioplasty. J Am Coll Cardiol 1999;34:1484–8.
2. Rankin JM, Spinelli JJ, Carere RG, et al. Improved clinical outcome after widespread use of coronary artery stenting in Canada. N Engl J Med 1999;341:1957–65.

Coenzyme Q10 as an Adjunctive Therapy in Patients With Congestive Heart Failure

Lack of effect from treatment with coenzyme Q10 in congestive heart failure is not an objective title or conclusion for the study by Watson et al. (1) in which the main limitation obviously is their sample size and its lack of study patients. Even so, the investigators state in their introduction that previous studies with coenzyme Q10 "lack credibility because of small sample sizes, lack of controls, etc."

The majority of the 27 study patients, who were not classified according to the New York Heart Association (NYHA), were seemingly at late-stage disease (mean length of symptoms 3.4 years). Mean patient age was 55 years, which is compatible with predominantly ischemic origin. This was also recently confirmed at an International Conference in Sydney, Australia—"Oxidative Pathways in Health and Disease"—in a lecture by one of the co-authors, Nicholas Bett (2). However, according to the Watson et al. (1) study, in the Patients' Demographics in Table 1, 77% of the patients were listed as having dilated cardiomyopathy. This is a patient clientele that is, at least partially, prone to respond either spontaneously or to medical intervention with subsequent improvement of myocardial function.

Conversely, it is well-known that changes—and not least improvements—in echocardiographic parameters of left ventricular (LV) function are minimal in late-stage disease, especially in heart failure due to ischemic heart disease. This is why the